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Original Article

Dermatoglyphic patterns as possible predictor of treatment resistance in schizophrenia

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ABSTRACT

Introduction: Dermatoglyphic patterns have been studied in schizophrenia and there is now evidence relating aspects of the dermatoglyphic profiles of the hands to schizophrenia.[1,2] But there has been no study on whether treatment resistance can be predicted from the dermatoglyphic profile of an individual. This study was done to determine whether abnormal neurodevelopmental markers were more frequent in schizophrenics non-responsive to routine treatment and whether treatment resistance can be predicted by studying the dermatoglyphic profile of an individual.

Methods: Finger and palm prints of 144 schizophrenic patients responsive to treatment and 44 schizophrenic patients non-responsive were taken. Finger print patterns, palmar patterns, palmar flexion creases, total ridge counts and atd angles were studied. The analysis was done using SPSS 11.0.

Results: The results showed there was increased pattern frequency in the I3 area of the left hand of non-responders and in the I4 area of the right hand of responders. atd angle was decreased in the left hand of non-responders. There was increased frequency of the Simian type I crease in male non-responders and Sydney line in female non-responders.

Discussion: Certain dermatoglyphic abnormalities occur more frequently in non-responders and with the help of these abnormalities, a schizophrenic patient likely to develop treatment resistance can be detected.

1. Introduction

Schizophrenia is a complex neurodevelopmental disorder conceptualized to be due to various insults of the brain with differing effects acting at different points of time.[1] Within a neurodevelopmental model of schizophrenia prenatal development deviations are implicated as early signs of increased risks for future illness. External markers of central nervous system maldevelopment may provide information regarding the nature and timing of prenatal disruptions among individuals with schizophrenia.[2] Finger and handprints are formed between the 11th and 24th weeks of foetal

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development and thereafter remain unchanged.[3] Skin and brain develop from the same ectoderm and cells migrate to the cortex at this time.[4] It is probable that an insult causing damage to one of these systems would damage the other, and there is now evidence relating aspects of the dermatoglyphic profiles of the hands to schizophrenia.[4,3] Many studies of people with schizophrenia have found abnormalities in brain structure like enlargement of the ventricles and decreased size or function of certain brain regions.[5] Developmental neurobiologists have found that schizophrenia may result when neurons form inappropriate connections during foetal development. These errors may lie dormant until puberty, when changes in the brain that occur normally during this critical stage of maturation interact adversely with the faulty connections. Therefore it is believed that schizophrenia may be, in part, a disorder of the development of brain. The rationale of studying dermatoglyphic features is derived from the fact that during their ontogeny, massive neural cell migration occurs in the brain, which is another ectodermal derivative. Thus, dermatoglyphic alterations in schizophrenia are markers of disrupted early development and contribute support to the developmental model of schizophrenia.[6,7] They show that an insult, whether genetic, environmental or both, occurred during early mid-gestation.[8,9] The relevance of dermatoglyphics is not to diagnose, but to prevent by predicting a disease; not for defining an existing disease, but to identify people with the genetic predisposition to develop certain diseases. Dermatoglyphic studies have been conducted in schizophrenia[10,11] but there has been no comparative study between the dermatoglyphic profiles of treatment responsive and treatment resistant schizophrenics.

The aims of the present study

1) To determine whether abnormal neurodevelopmental markers were more frequent in schizophrenics non-responsive to treatment than those responsive.
2) Whether treatment resistance can be predicted by studying the dermatoglyphic profile of an individual.

2. Materials and methods

The study sample consisted mainly of outpatients and some inpatients between 18 and 60 years of age suffering from schizophrenia. 144 of them (86 males and 58 females) were schizophrenics responding to treatment and 44 (35 males and 9 females) were schizophrenics not responding to routine treatment. The patients not responding to treatment were determined by the fact that they were put on clozapine due to drug resistance or other indications like tardive dyskinesia. The patients belonged to the F20 category in ICD-10 classification. Patients were considered treatment resistant using the modified Kane's criteria. All subjects gave informed consent. The socio demographic and clinical data and details of psychiatric history were obtained from the patient or from their medical records. Finger and palm prints were obtained by the ink and pad method[7] (Fig. 6a–e) and the analysis of qualitative and quantitative dermatoglyphic features was undertaken, with the aid of a magnifying glass.[7,8]

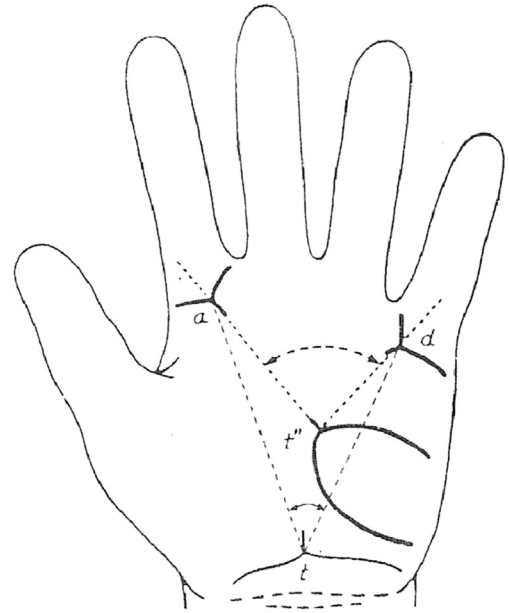

Fig. 1 – atd angle measurement.

2.1. *Inclusion criteria*

All inpatients and outpatients attending the Psychiatry OPD diagnosed to have schizophrenia (F20-category) by ICD-10 criteria.

2.2. *Exclusion criteria*

Patients with following conditions were not included in the study:

1) Obstetrical complications

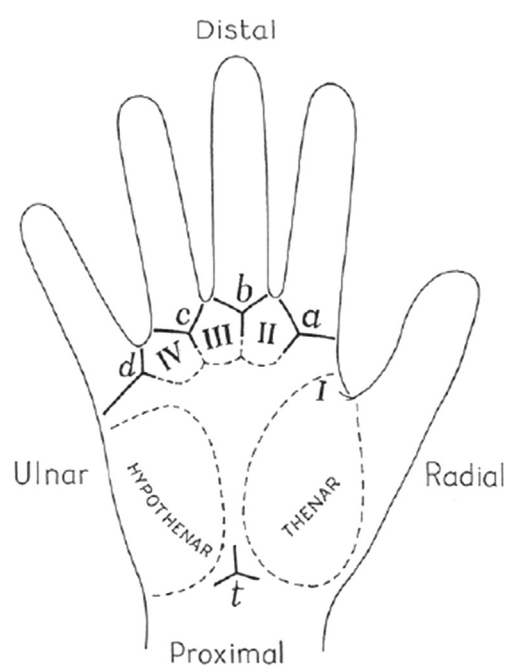

Fig. 2 – Dermatoglyphic palmar pattern areas.

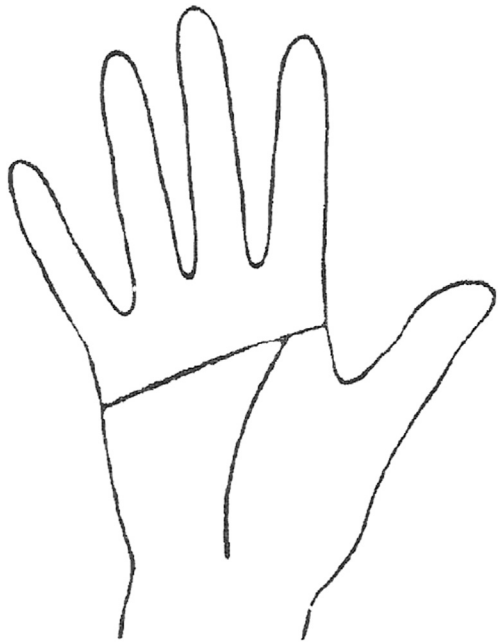

Fig. 3 – Single transverse crease or simian crease.

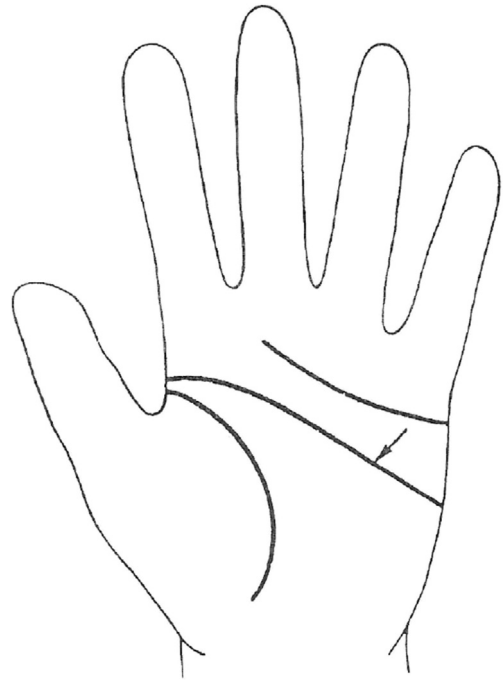

Fig. 5 – Sydney line.

2) Untreated psychosis
3) Vulnerability to tardive dyskinesia due to other antipsychotics.

Materials used were:

1) Kores finger print ink
2) Ink roller
3) Slab
4) Paper for taking the prints
5) Soap and water for removing the ink from the hands

After the finger and palm prints were obtained they were analysed. The qualitative analysis did include finger print patterns (Fig. 6a–d), patterns in palmar interdigital areas (Fig. 2) and palmar flexion creases (Figs. 3–5).

The quantitative analysis include total and absolute finger ridge counts, a–b ridge count (Fig. 6e) and atd angle (Fig. 1).

2.3. *Statistical analysis*

Chi-square/Fischer's exact tests were used for group comparisons.

Independent t-test (for normal data) and Mann–Whitney test (for non-normal data) were performed to compare the mean scores between treatment responders and non-responders.

Pearson correlation analysis were done to assess the relationship between treatment responders and non-responders with respect to the ridge counts.

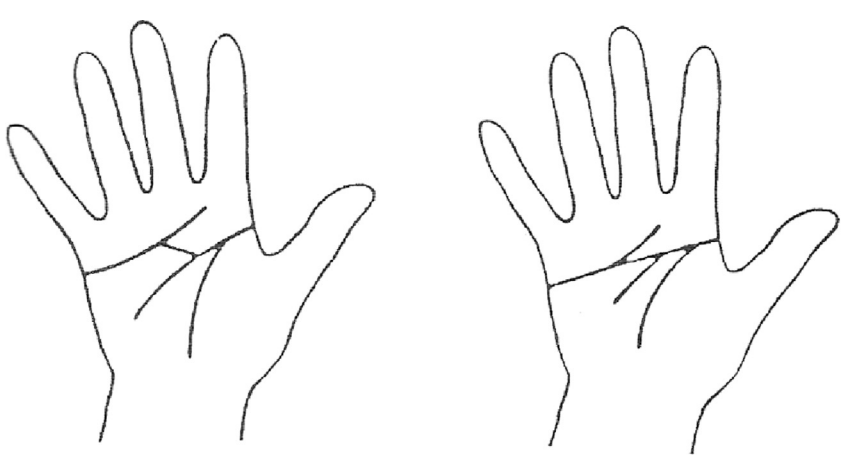

Fig. 4 – Simian transitional type 1 & type 11.

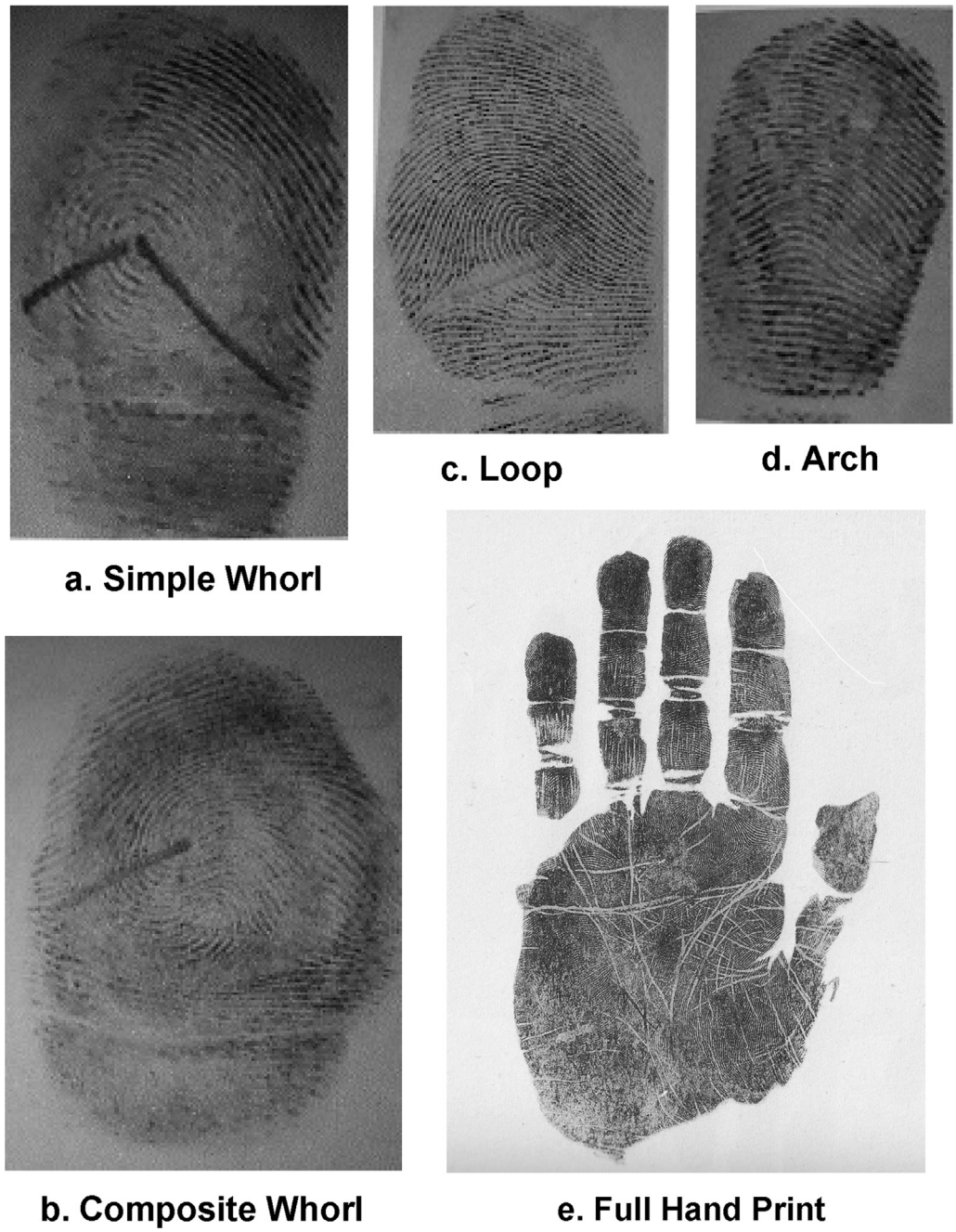

Fig. 6 – (a) Simple whorl; (b) Composite whorl; (c) Loop; (d) Arch; (e) Full hand print.

All the analysis were carried out using SPSS software Version. 11.0.

3. Results

The results of the analysis are presented in Tables 1–7.

3.1. *Palm print patterns*

From Tables 1 and 2 it is seen that there is a statistically significant difference between the diagnostic groups for the palmar patterns (Fig. 2) in the interdigital areas I_3, I_4 and hypothenar areas.

3.2. *Palmar flexion creases*

It is seen from Tables 3 and 4 that there is a statistically significant difference between the diagnostic groups for the abnormal palmar flexion creases. Simian transitional type I crease (Fig. 4) is significantly more frequent in male non-responders and Sydney line in female non-responders.

Table 1 – Comparison of the frequency of palmar patterns occurring in interdigital and hypothenar areas of the right hand of treatment responders with treatment non-responders (Fig. 2).

Pattern area	Treatment responders		Treatment non-responders		x^2	Significance
	$n = 144$	%	$n = 44$	%		
Thenar + I_1	26	18.1	6	13.6	0.466	0.466
I_2	97	67.4	24	54.5	2.413	0.118
I_3	84	58.3	23	52.3	0.504	0.482
I_4	125	86.8	32	72.7	4.850	0.027*
Hypothenar	44	30.6	13	29.5	0.016	0.890

*P < 0.05.
I = Interdigital area.

3.3. *Total ridge count*

There is a higher total ridge count in treatment responders but the differences are not significant (Table 5).

3.4. *atd angle*

The mean atd angle (Fig. 1) is less in the left hand of treatment non-responders and this difference is statistically significant (Table 5).

3.5. *Finger print patterns*

There was no significant difference in the finger print patterns between the diagnostic groups (Table 6), (Fig. 6a–d).

3.6. *a–b ridge count*

There is no significant correlation in the a–b ridge count in male and female non-responders (Table 7), (Fig. 6e).

Table 2 – Comparison of the frequency of palmar patterns occurring in interdigital and hypothenar areas of the left hand of treatment responders with treatment non-responders (Fig. 2).

Pattern area	Treatment responders		Treatment non-responders		x^2	Significance
	$n = 144$	%	$n = 44$	%		
Thenar + I_1	43	29.9	12	27.3	0.109	0.740
I_2	125	86.8	35	79.5	1.401	0.234
I_3	66	45.8	28	63.6	4.272	0.039*
I_4	104	72.2	34	77.3	0.440	0.503
Hypothenar	28	19.4	14	31.8	37.525	0.000*

*P < 0.05.

Table 3 – Comparison of the frequency of abnormal palmar flexion creases of treatment responders with treatment non-responders (males) (Figs. 3–5).

	Treatment responders (N = 86)		Treatment non-responders (N = 35)		x^2	Significance
	n	%	n	%		
Right hand						
ST – I	19	22.2	20	57.1	13.990	0.000*
ST – II	1	1.2	–	–	–	–
SL	8	9.3	5	14.3	0.644	0.421
SFC	1	1.2	1	2.9	0.439	0.511
Normal flexion creases	**57**	**66.3**	**9**	**25.7**	–	**0.000***
Left hand						
ST – I	16	18.6	17	48.6	13.265	0.000*
ST – II	2	2.3	–	–	–	–
SL	7	8.1	5	14.3	1.051	0.300
SFC	3	3.5	–	–	–	–
Normal flexion creases	**58**	**67.4**	**13**	**37.1**	–	**0.002***

*P < 0.05.
n = No. of observations made.
N = No. of patients observed.
ST – I = Simian transitional type I.
ST – II = Simian transitional type II.
SL = Sydney line.
SFC = Single flexion crease.

Table 4 – Comparison of the frequency of abnormal palmar flexion creases of treatment responders with treatment non-responders (females) (Figs. 3–5).

	Treatment responders (N = 58)		Treatment non-responders (N = 9)		x^2	Significance
	n	%	*n*	%		
Right hand						
ST – I	7	12.1	3	33.3	2.774	0.097
ST – II	–	–	–	–	–	–
SL	6	10.3	4	44.4	7.134	0.007*
SFC	–	–	–	–	–	–
Normal flexion creases	**45**	**77.6**	**2**	**22.2**	–	**0.000***
Left hand						
ST – I	9	15.5	3	33.3	1.682	0.194
ST – II	–	–	–	–	–	–
SL	8	13.8	4	44.4	4.978	0.025*
SFC	–	–	–	–	–	–
Normal flexion creases	**41**	**70.7**	**2**	**22.2**	–	**0.004***

*P < 0.05
n = No. of observations made
N = No. of patients observed
ST – I = Simian transitional type I
ST – II = Simian transitional type II
SL = Sydney line
SFC = Single flexion crease.

4. Discussion

The dermatoglyphic features analysed in the present study point to certain significant differences between treatment responders and non-responders.

Schizophrenics are found to exhibit simpler fingertip patterns.[12,13] In the present study, there is no difference in the frequency of the fingertip patterns (Fig. 6a–d) between the treatment responders and non-responders. The ulnar loops (Fig. 6c) occurred more frequently than radial loops in both groups.

The frequency of the hypothenar pattern was higher in the left hand of treatment non-responders than in treatment responders. Usually, in most individuals there is no pattern in the thenar area, but the ridges follow a mild course around the

Table 5 – Comparison of the quantitative dermatoglyphic features of treatment responders with treatment non-responders (Fig. 1).

	Mean	SD	Significance of difference
Total ridge count			
Treatment responders (N = 144)	184.4	59.7	t = 0.727
Treatment non-responders (N = 44)	177.1	53.2	P = 0.470
'atd angle'			
Right hand			
Treatment responders (N = 144)	38.96	5.07	t = 0.447
Treatment non-responders (N = 44)	38.56	5.56	P = 0.645
Left hand			
Treatment responders (N = 144)	40.00	6.79	t = 2.878
Treatment non-responders (N = 44)	36.91	3.83	P = 0.004*

*P < 0.05.

Table 6 – Frequency of loops and whorls (Fig. 6a–c).

Pattern	Treatment responders (N = 144)		Treatment non-responders (N = 44)		P value
	n	%	*n*	%	
Loop					
Ulnar	805	55.63	248	55.68	0.000*
Radial	27	1.84	8	1.80	
Whorls					
True	397	27.26	122	27.60	0.893
Composite	101	6.91	32	7.23	

*P < 0.05.

Table 7 – a–b ridge counts of the right & left hands in treatment responders & non-responders (Fig. 6e).

	Responders (N = 86)		Non-responders (N = 35)		P value
	Mean	SD	Mean	SD	
a–b ridge count – females					
Right	46.6	9.9	49.4	5.3	0.213
Left	47.9	9.4	45.0	6.5	0.372
a–b ridge count – males					
Right	46.9	11.1	43.9	10.3	0.179
Left	46.6	8.7	46.6	9.3	0.989

base of the thumb. Patterns when present are more often loops.[14–16] The pattern frequency in the thenar/I1 area showed no significant difference between the two groups.

The pattern frequency in the I2 area showed no significant difference between the two groups. The pattern frequency in the I3 area (Fig. 2) of the left hand of treatment non-responders was higher than in the responders. Fearon et al, (2001)[3] in their study of 148 schizophrenic patients responsive to treatment, found decreased frequency of patterns in the right fourth interdigital area. In the present study, the pattern frequency in the I4 area of the right hand of treatment responders was higher than in the non-responders.

Fananas et al, (1996)[12] and Davis and Bracha (1996)[17] found no significant difference in total finger ridge count in the sample they studied of schizophrenics who were responsive to treatment. Turek (1990)[13] reported a decrease in a large sample of responsive schizophrenic patients. In the present study, there is no difference in TFRC between the study groups. TFRC appears to be under relatively strong genetic control and little influenced by environmental events.[18]

Bramon, et al (2005)[19] confirmed the presence of significant yet mild ABRC (a–b ridge count) (Fig. 6e) reductions in schizophrenia. These represent a subtle deviance from the norm and possibly in those who suffered early developmental insults.

Shana Golembo-Smith, et al (2012)[2] study indicated significant but small effects for total finger ridge count and total a–b ridge count, with lower counts among individuals with schizophrenia relative to controls.

Our study confirms that certain abnormal neurodevelopmental markers were more frequent in schizophrenics non-responsive to routine treatment and by studying the dermatoglyphic profile, a patient likely to develop treatment resistance can be detected at an early stage.

Conflicts of interest

All authors have none to declare.

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